BULLETIN OF
THE NEW YORK ACADEMY
OF MEDICINE

VOL. 39, NO. 2 FEBRUARY 1963

MANAGEMENT OF NEUROGENIC VESICAL DYSFUNCTION*

HERBERT S. TALBOT

Chief of Staff and Chief of Urology,
Veterans Administration Hospital, West Roxbury, Massachusetts;
Clinical Associate in Surgery, Harvard Medical School;
Chairman, World Committee on Spinal Paraplegia

THE neural mechanism that controls micturition is complex and its components widely distributed. Theoretically, it might be possible to consider the effect of any conceivable lesion upon bladder activity, but clinical experience embraces only a relatively small group of neurological disorders regularly associated with vesical dysfunction. Even these, however, are too many to be within the scope of an essay of reasonable length. In confining myself to a single type, I remain securely within the limits of my own experience and take advantage of the common usage by which the term "neurogenic bladder" is generally associated with injury or disease of the spinal cord.

In order to validate the system of management to be presented, it is necessary to begin with a brief review of the concept of the physiology of micturition upon which it is founded. This will be offered in

* Presented as part of a program on *The Neurogenic Bladder,* held at the meeting of the Section on Urology, The New York Academy of Medicine, January 17, 1962.

From the Spinal Cord Injury Service and the Urological Section of the Surgical Service, Veterans Administration Hospital, West Roxbury, Massachusetts.

rather general terms, omitting details which are readily available elsewhere.[1] There will be no attempt to set up a classification of dysfunction. Such classifications tend to become Procrustean; we fit cases to them instead of them to cases and run the risk of discovering, one day, that we are treating categories rather than patients. There are, of course, patterns of dysfunction which are reasonably consistent and demand recognition. The correlation of such patterns with known lesions provides valuable clues to the physiological mechanisms involved. The dysfunction as it presents itself clinically, however, is seldom the direct and immediate result of a specific lesion. Rather, it is a stage in a sequence of events in which function and structure react upon and interact with each other, and are further influenced by secondary agents. Thus, for example, a patient with dysfunction of micturition associated with spinal cord injury may exhibit the combined result of altered neural activity, hypertrophy of the bladder wall in response to such alteration, infection of the bladder as a result of stasis, and fibrosis as a result of infection, not to mention calculus formation, possible effects of instrumental trauma, and the considerable influence of the patient's personality. Such a case can be described as one of neurogenic vesical dysfunction. It is that, of course, but with an overlay of a variety of secondary dysfunctions. Any consideration of the physiology of micturition, if it is to be clinically significant, must include the recognition and evaluation of all these factors.

This discussion is offered not as a fully developed theory of how the mechanism of micturition operates but as a concept of how it may operate, consistent with observed facts, with the general laws of physiology, and requiring no exceptions to, nor specializations of those laws. It has provided a basis for reasonably accurate predictions which can be translated into a satisfactory system of clinical management. Fundamentally a spinal reflex activity, the primitive character of micturition has been altered by modifications and controls mediated through centers above the cord.[2] When these are lost, what remains is no longer adequate for the normal demands of daily life unless they can be re-established or replaced. The urinary bladder has two functions: it must hold urine and expel it, and both of these activities must be adapted to meet varying demands. This dichotomy is subserved by a single peripheral apparatus with a complex synaptic background. The musculature of the bladder wall is arranged in an intricate feltwork.[3]

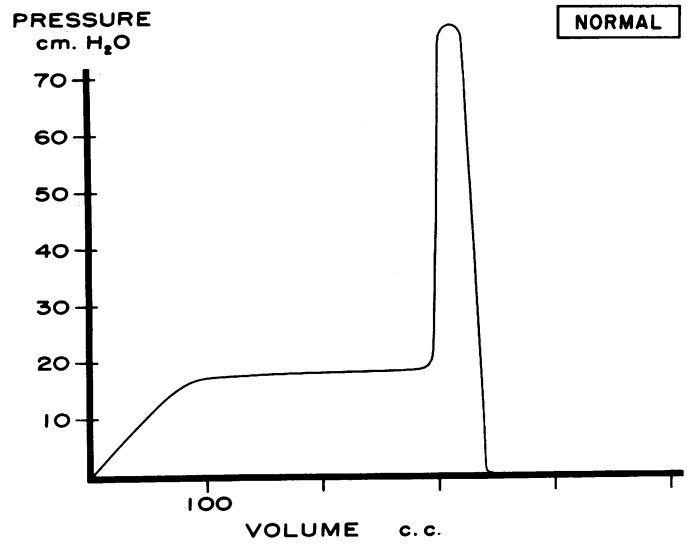

Fig. 1. A conventionalized curve of the alterations in bladder pressure during filling and emptying of a normal bladder, showing maintenance of a constant rather low level until the emptying contraction begins, with a fall to zero as emptying is completed.

Although smooth muscle is capable of contracting in response to stretch without nervous stimulation, it is apparent that purely myogenic activity would result only in disconnected contractions, occurring in various parts of the wall and incapable of producing an efficient detrusor action. The neural component of the mechanism provides stretch receptors which feed afferent impulses into a reflex arc. In the sacral spinal centers these are translated into appropriate effector responses. The bladder, while filling, is thus able to adapt its configuration to its content very precisely. Within a wide range, this content is held in a gentle grip, and the mean intravesical pressure remains almost constant. One of the most impressive characteristics of normal micturition is the low range of pressures within which the function operates. When the critical point of distension is reached, the so-called emptying contraction is provoked. This is actually a series of events, with muscle bundles in various parts of the bladder contracting in such order as to produce an efficient sustained propulsive force. At the same time, the bladder neck is drawn open, probably as a result of the arrangement of the bundles in that region. The tonicity of the bladder neck and the proximal portion of the posterior urethra, which together

constitute the functional internal sphincter, is capable of withstanding the low pressures at which urine is usually carried in the bladder. When these pressures increase sharply, as during the emptying contraction, they give way unless there is organic obstruction or untimely contraction. Observed fluoroscopically, the whole act is seen to proceed with remarkable smoothness.[4]

The detrusor is supplied by parasympathetic fibers that pass through the pelvic nerves. Sympathetic fibers run to the trigonal area, the internal sphincter and the genital passages. The two systems are reciprocal rather than antagonistic. Interruption of the sympathetics has no apparent effect on micturition but may interfere with ejaculation. During the latter act, sympathetic impulses cause contraction of the internal sphincter, preventing the escape of urine and the reflux of semen. Simultaneously, there appears to be a secondary reflex inhibition of the detrusor. The external sphincter mechanism is provided mainly by the deep transverse perineal and other striated muscles within the urogenital diaphragm, innervated by the pudendal nerve. During their voluntary contraction, there is again reflex inhibition of the detrusor, according to some observers. Striated muscle fibers have been described as extending into the smooth muscle structure of the bladder neck, particularly its anterior portion.[5] Involuntary spastic contraction of any of these striated muscle elements, as seen in some patients with spinal cord lesions, may obstruct micturition.[6]

In the human after infancy, the spinal reflex mechanism operates subject to controls imposed upon it from higher centers, operating through pathways in the spinal cord. This is suggested by the fact that, following transection of the spinal cord, there is usually a tendency to hypertonicity of the bladder muscle. Reference has already been made to the generally low pressures normally operative. The tonic adaptability of the bladder may be mediated chiefly within the spinal reflex centers, but deprived of the higher controls, it is no longer so precise. Another supra-segmental control which, like the tonic factor, seems to be derived from centers in the mid-brain, brain stem, and possibly the cerebellum, is best described as an integrative action. By the indirect evidence of the effect of its withdrawal, this causes the contraction of the detrusor muscle to follow an orderly pattern as it sweeps over the bladder wall to produce complete emptying with minimal effort. Lacking this integrating modulation, the blad-

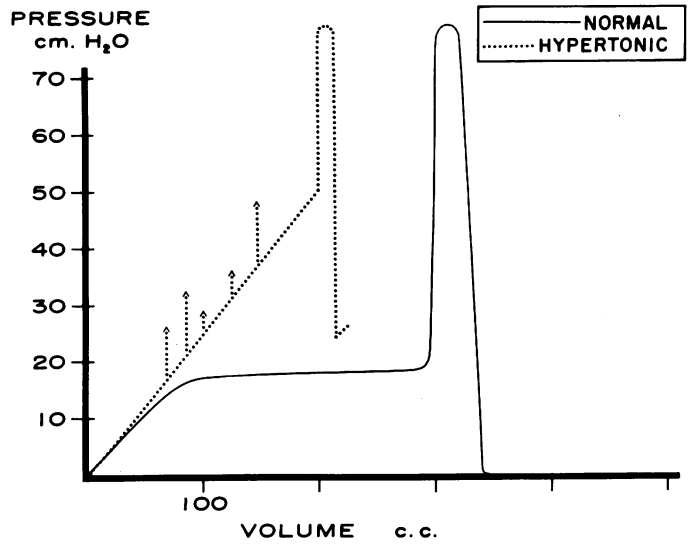

Fig. 2. The normal curve contrasted with that characteristically seen in the hypertonic bladder of neurogenic vesical dysfunction. The latter shows a steep gradient and frequent ineffectual contractions. The "emptying" contraction is inadequate, and leaves residual urine; the pressure, in consequence, never falls to zero.

der still contracts vigorously but less efficiently; taken together with the hypertonicity, this results in the apparent paradox of a bladder with a powerful hypertrophied musculature, contracting frequently, yet unable to expel its contents.[7]

Of great importance are the cortical centers which mediate conscious awareness of the state of the viscus and voluntary inhibition of detrusor contraction. This is the normal mechanism of voluntary control of micturition, which is lost after transection of the spinal cord. An accessory means of control is provided by the external sphincter. Under ordinary circumstances this need not be used to prevent voiding except when the bladder is voluntarily over-distended, but it is probably employed quite regularly to effect a sharp cut-off at the end of the act; it can also be successfully invoked to interrupt micturition after it has begun.

The afferent components of micturition have had more attention in recent years. This is an important development since a neuromuscular function is basically a response to the streaming impulses which initiate and control it. These, in turn, are modified during

activity, be it normal or abnormal, creating an effective feed-back mechanism. The stretch receptors, already mentioned, are specific in function. The adaptation of the bladder wall to a variable content and its ultimate emptying contraction are responses to this stream of impulses. Other receptors are located in the mucosal, rather than the muscular layer, and these pick up other stimuli, particularly those resulting from inflammatory or irritative processes. By analogy, they may be considered exteroceptive and the stretch receptors proprioceptive.[8] They react readily to cold, which permits of an easy and harmless method of experimental stimulation. These receptors are responsible for the familiar hyperactivity seen in the presence of infection or calculus. Although the usual response to their abnormal stimulation is hyperactivity, it may be that, in certain circumstances, they provoke sphincteric contraction and so impede micturition. It has been found, for example, that certain patients with neurogenic vesical dysfunction, who have previously been unable to void, can empty their bladders effectively after the instillation of a topical local anesthetic.

The importance of vesical dysfunction in the etiology and pathogenesis of upper urinary infection has led to investigation of the relationship of ureteral function to micturition. There is evidence that the activity of the lower segment of the ureter constitutes, in effect, a dynamic ureterovesical valve. Anatomically, its wall is the thickest of any portion, and its outer muscular layer merges indistinguishably with the musculature of the bladder. The lower third of the ureter, moreover, is supplied with parasympathetic ganglia, identical to those in the adjacent portions of the bladder wall. Thus, there is strong anatomical presumption of an intimate functional relationship between the bladder and the lower ureteral segment. Other observations indicate that the lower end of the ureter has a task somewhat different from that of the upper end, and that the integration of tonic and peristaltic activity is such as normally to protect the nephron from the high basic pressures which arise at the lower end of the ureter when it is operating to handle large quantities of urine, or against elevated intravesical pressures.[9] Fluoroscopic studies, including recent cinefluorographic investigations, have demonstrated that the lower ureter does, in fact, contract during or immediately after an emptying contraction of the bladder. Again, fluoroscopically, it has been possible to

demonstrate that in some patients with vesico-ureteral reflux, the mechanism remains sufficiently well compensated to operate in approximately normal fashion, and the ureter empties during voiding. When such compensation is no longer possible, vesico-ureteral reflux becomes more marked during micturition. Clinically, the distinction is very important. Reverse peristalsis, long since observed in the presence of mechanical obstruction, has more recently been seen to occur also in this functional type.[10]

So brief a discussion as this runs the usual risks of oversimplification. It is obvious that there are gaps in our understanding which it is better to admit than to fill in with unfounded speculation. Yet from this clinical consideration of the physiology of micturition, the outlines of a pattern emerge which not only indicate avenues for further investigation but form a basis for rational management of abnormal function. It is of some value to know that certain events do occur in certain circumstances, even though we cannot yet say how or why. If we think of function as the response of the organism to a given set of stimuli under given conditions, we may define dysfunction as the altered response which occurs when the stimuli or the conditions have been altered. But these are often within our power to change, if we can but decide what changes are necessary.

Two aims should be paramount: the development of a satisfactory micturitional function and the preservation of the integrity and function of the upper urinary tract. Far from being incompatible, the two represent synergistic efforts; a healthy bladder with at least a semblance of normal function is a powerful safeguard if not absolute insurance against renal disease. If, in addition, the formation of calculi because of disturbed calcium metabolism can be prevented, it should be possible to protect the patient from the disastrous train of events that has led so many to invalidism and death.

As applied to an individual patient, the goal of treatment is to achieve:

1. Urethral voiding.
2. Adequate capacity.
3. Avoidance of residual urine.
4. Control, of a sort.

The reciprocal relationship between function and structure and the likelihood of secondary changes such as those resulting from infection

have already been mentioned. Such structural alterations may become severe and irreversible so that, apart from the neural deficit, they constitute an impediment to any restoration of useful function. The hazard is present at the very beginning and must be met. During spinal shock the bladder temporarily loses its emptying function; that is, it no longer responds to an appropriate degree of distension by going into a forceful and efficient contraction. It does not become completely atonic, however, as was once thought to be the case. Cystometric studies done immediately after spinal cord injury show a relatively normal tonus, but atonicity will soon appear as a result of overdistension.[11] Avoidance of such overdistension is the imperative rule of early treatment, and the best way to accomplish this is by continuous urethral catheter drainage. It is true that overflow voiding will usually develop, but this leaves a large residual which soon becomes infected, adding further insult to the injury already done the bladder wall.[12] Manual expression by the Crédé maneuver is traumatizing and seldom achieves complete emptying. Intermittent catheterization may postpone the infection that accompanies the indwelling catheter, but not for long enough to carry the patient to a stage of satisfactory function; if done often enough to be effective it involves what seems to be an excessive amount of manipulation. Suprapubic cystostomy is justified when, as in some military casualties, a proper catheter hygiene cannot be maintained. It is preferable to allowing the bladder to remain undrained, but may delay the resumption of good reflex function. There are, however, instances in which it should be considered as a definitive method of permanent drainage, notably when there is irreparable urethral obstruction, or for those quadriplegic patients who cannot accomplish the manual maneuvers incident to voiding. Perineal urethrostomy has no advantage over suprapubic drainage, and is much more difficult to manage in a patient prone to pressure ulcers and, perhaps, incontinent of feces.

The disadvantages of the indwelling urethral catheter can be, and have been, over-emphasized.[13, 14] Taken all together, they add up to a good deal less than the danger of infection in an inadequately drained bladder. Furthermore, by preserving structural integrity, early drainage hastens the day when the process of re-training will have been achieved and artificial drainage abandoned. Needless to say, precise aseptic technics and a meticulous urethral toilet are essential. A small Foley catheter, size 16-F, should be used, and there is much to be said for institut-

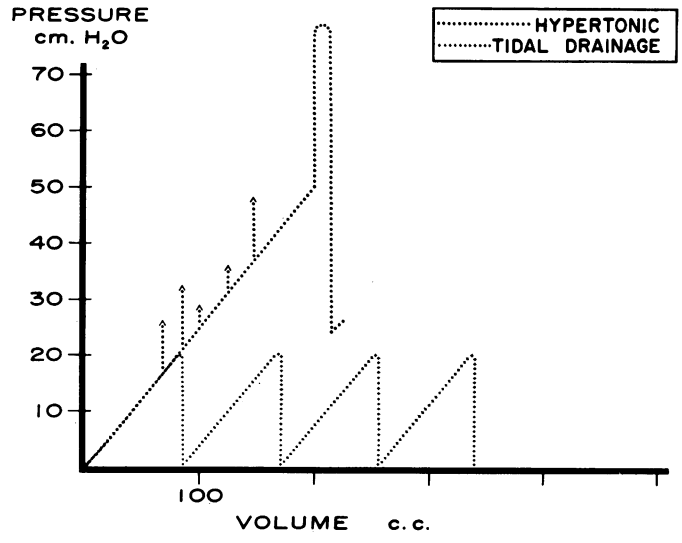

Fig. 3. The curve of hypertonic dysfunction contrasted with that representing activity during tidal drainage, during which pathological ranges of pressure are avoided and the bladder empties periodically. (The brief period of negative pressure during siphonage is not shown).

ing tidal drainage at this stage. With the siphon loop set at a low level this provides the hygienic advantage of frequent irrigation within a closed system, and the rhythmic filling and emptying of the bladder is preferable to the continuously contracted state of an empty viscus.

As the patient emerges from spinal shock, secure in the advantage of adequate drainage, reflex activity below the level of the lesion is resumed. The bladder, although without its higher controls, will now fill and empty with variable regularity. In this situation it is sometimes described as "automatic" but the term is so loosely used as to be generally meaningless. In any event, the type of function just mentioned, whatever it may be called, is unsatisfactory. Such a bladder tends to become hypertonic; its emptying efforts are inefficient and residual urine accumulates. There is, of course, no voluntary control. As time passes, the wall hypertrophies and hypertonicity increases. The combination of elevated intravesical pressure and continuing cystitis opens the way to ascending infection.[9, 10] In such worsening circumstances hope of salvaging bladder function may be abandoned and the patient put on permanent suprapubic or urethral catheter drainage, or subjected to some type of procedure for extra-vesical diversion. But such a situa-

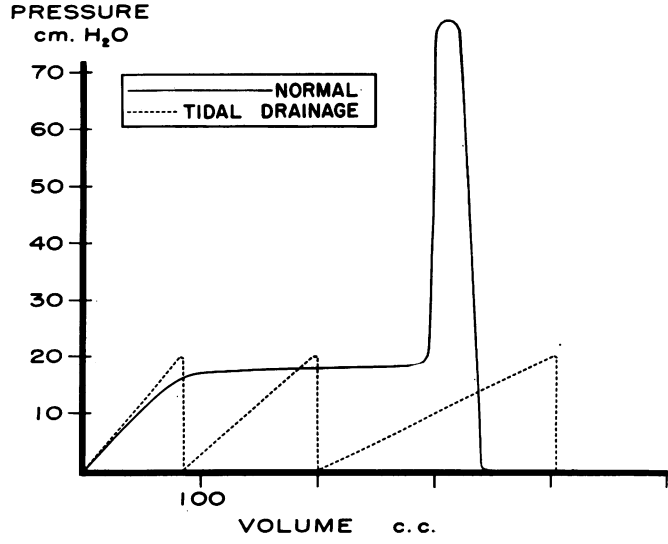

Fig. 4. Pattern of tidal drainage as conditioning progresses, showing the bladder gradually acquiring a greater capacity within the same range of pressure, and indicating the eventual possibility of approximation to the normal pattern.

tion can almost always be avoided; when encountered, it may yet be amenable to functional restoration.

The management here suggested is based upon the fact that reflex activity can be modified by conditioning directed toward greater consistency and efficiency. Tidal drainage is one method of applying such conditioning—not the only method, but a good one, and particularly applicable when a large group of patients is involved. Many types of apparatus are available, differing in design, appearance, and cost, but all necessarily based upon the same fundamental physical principle of siphonage. On the assumption that the best machine is the simplest that will do the job, my own preference is for a home-made arrangement of glass and rubber tubing, easily cleansed and sterilized, and very inexpensive. It is, nonetheless, a piece of machinery; like any machine, it cannot think for itself, nor should it be expected to perform work for which it was not designed.

Although its use frequently leads to an increase in the capacity of the bladder, it is wrong to think of tidal drainage as a means for "stretching" that viscus. Its effect, including the increase in capacity, derives not from forceful hydraulic distension, which is undesirable,

but, it is conjectured, by conditioning the neuromuscular mechanism to conduct its operations within a more nearly normal range of intravesical pressures. As already mentioned, it may be used to advantage during the period of spinal shock, with the siphon loop set at a level of perhaps 6 cm. above the symphysis pubis. As the reflex emptying contractions return, the loop is gradually elevated, at the rate of 2 cm. every two or three days, until it reaches a level of 16 to 20 cm. There it is held for about two weeks if the patient is one who has come to treatment immediately after injury. Tidal drainage is then discontinued and the catheter clamped off, the clamp being opened at intervals corresponding to those at which siphonage occurred, with allowance for the bladder being filled now from the kidneys only. After a few days of this, the catheter is removed for a trial at voiding, the patient being instructed to void at regular timed intervals. These should be rather brief at first—30 to 45 minutes—since it may take the internal sphincter some days to regain continence after removal of the indwelling catheter. It is also desirable, during this phase, to reduce temporarily the high fluid intake usually prescribed for these patients. The interval between voidings is gradually extended to 2 or 2½ hours which is probably optimal.

Many patients retain various types of sensation, some of it simulating normal.[15, 16] Frequently, however, the patient who feels what he thinks is a desire to void is actually beginning to void. Only rarely is this residual sensation as reliable as timed habit. Those with cervical or high thoracic lesions (above T-3) are subject to automatic hyperreflexia with bowel or bladder distension. Some of the symptoms so produced, headache, flushing, or goose pimples, may be more dependable signals than the sensations occurring with lower levels of injury. But these phenomena, which include paroxysmal hypertension, may be dangerous, and treatment should be undertaken to prevent them.

Initiation of voiding is usually accomplished quite readily by abdominal straining, the application of manual pressure, or brisk tapping of the abdominal wall. When this type of management is started early, before the onset of any complicating factors, and if the patient is in reasonably good general condition, the whole process need take no longer than six or eight weeks, and a good result is the rule. The bladder has never been overdistended, it has been filling and emptying in physiologic fashion, and the intravesical pressure, limited by the level

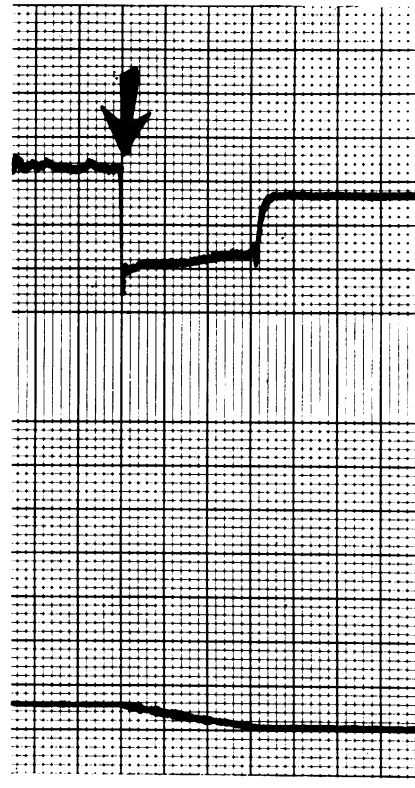

Fig. 5. Portion of tracing made during continuous recording of bladder activity during tidal drainage. The upper tracing records pressure; the lower records volume, the excursion toward the bottom of the page representing the evacuation of fluid from the bladder. In this instance, there is no suggestion of any bladder activity prior to the beginning of siphonage.

of the siphon loop, has never been excessive. The patient now has a consistent and efficient micturitional function. Although he can no longer inhibit reflex voiding, he can anticipate it and thus achieve a type of control. For some, this is all but perfect. Others experience stress incontinence on straining, sneezing, or the like. This is particularly the case among those who neglect to keep track of the interval between voidings or who try to prolong it unduly. For protection against wetting, they may use a condom-coupled urinal. There is no harm in this, with proper hygienic care, but it is a supplement to, rather than a substitute for good habit training. Of all our patients, about 70 to 75 per cent get along without catheter after discharge

from the hospital. Perhaps 40 per cent of these use such a device, many of them part of the time only.

All too frequently, the patient is not seen as early in his course as might be desired. At the time of his admission, if he has been voiding with a so-called "automatic bladder", it will usually be hypertonic and the intravesical pressures already elevated. If a catheter has been in place for constant drainage, the bladder will probably be contracted, its wall showing fibrotic changes as well as trabeculation, and its capacity diminished. The role of tidal drainage at this stage must be corrective rather than preventive. There may be sufficient intolerance to the inflow of fluid from the apparatus to require the rate to be reduced below the customary 60 drops per minute, while the siphon loop must be set higher than usual to permit accumulation of a reasonable quantity before siphonage occurs. It may be impossible to employ tidal drainage without preliminary recourse to some means of controlling hypertonicity. Usually, however, suitable adjustments can be made, after consideration of the cystometric findings and, perhaps, a period of trial and error. If successful, it imposes upon the pattern of dysfunction a new pattern, calling for responses at more nearly normal intravesical pressures and eliminating the pathologically high range. It takes longer, with a late start, and the outcome is less certain, but the results have in general been encouraging.

Empirical experience suggests, and it is reasonable to theorize, that the mechanism invoked is indeed one of conditioning. Limitation of this term to processes involving the cerebral cortex is no longer valid. Menzies has pointed out that, "It is not justifiable to conclude cavalierly that all conditioning follows the principles of conditioned salivation in dogs. The experimental evidence already available indicates that this is not the case, but rather that different organisms and different response systems within the same organism present their own peculiarities of conditioning." [17]

When the contents of the bladder are removed by siphonage, its wall contracts, adapting its configuration to an empty state. It is logical to infer that after the detrusor has done this many times, always at the same level of pressure, it will have been conditioned to do so when this pressure is reached even though siphonage does not occur. Demonstration of such contraction would support the conditioning theory. With this in mind, Dr. O'Hare, one of my associates, and his technical as-

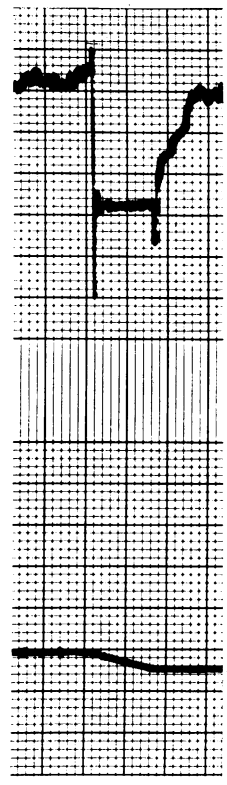

Fig. 6. Tracing similar to Fig. 5 from another patient, showing an increase in bladder pressure, which represents contraction, immediately prior to or at the instant of the beginning of siphonage. The amount evacuated in this instance was about 250 cc. The pressure shows a transitory negative phase during siphonage; then, during refilling, promptly rises to a physiological level.

sistant, Mr. Godin, have devised a method of continuous recording of the activity of the bladder on tidal drainage. On some of their tracings, the fall of pressure when the bladder is emptied is clearly preceded by a preliminary rise which must represent contraction of the detrusor since there is nothing in the operation of the apparatus to produce it. In recordings of other patients no such contraction is observed, indicating that conditioning has probably not yet taken place. The number of studies thus far undertaken is too small to constitute anything like proof, but the evidence is suggestive.

The fluid used during tidal drainage is usually a bland bacteriostatic solution such as 1:20,000 Zephiran. Occasionally, one of the buffering

solutions may be indicated. In the case of an extremely intolerant bladder, physiologic saline solution is the choice. Catheter and apparatus are changed at least once a week, more often if there is heavy sediment in the urine. The waste jug is replaced every eight hours. It is desirable to instruct the patient as to what is being attempted, and arrange the set-up so that he can watch and report on its operation.

The foregoing is chiefly applicable to cases in which the level of the spinal cord lesion is cephalad to the sacral segments where the reflex centers for micturition are located. Bors has called these upper motor neuron lesions in contrast to lower motor neuron lesions resulting from injury to the conus or cauda equina. The latter, not infrequent among military casualties, are relatively rare in civilian injuries. Obviously, if the reflex center has been destroyed or the arc interrupted, the conditioning effect of tidal drainage is no longer available. The hygienic advantage remains, however, as well as protection against contracture. Its use, therefore, is justified, particularly during early treatment. The postganglionic fibers arising from the intramural ganglia of the bladder are apparently incapable of provoking emptying contractions, but maintenance of healthy musculature is still desirable. Some patients with lower motor neuron lesions ultimately manage without catheters, initiating and maintaining detrusor contraction by a sort of Crédé maneuver, but in my experience the results in these cases are generally less satisfactory than in the other group.

In a series of nearly a thousand patients, those who have exhibited obstacles and impediments to the success of this program comprise a heterogeneous and formidable group. Sometimes—and this is particularly provoking—the reason for failure remains unknown. Sometimes it is sufficiently apparent but we don't seem to be able to do anything about it—and this is, perhaps, more provoking still. Over the years, however, a number of adverse factors have emerged which can be recognized and, with variable success, brought under control. One of the most frequent is obstruction at the bladder outlet. Relatively few of our patients have yet reached the age of prostatic hypertrophy; that is a problem we anticipate. The obstruction usually arises from the fact of the musculature around the orifice sharing in the general hypertrophy of the bladder wall, to which there is always added an element of fibrosis. The essential feature, in fact, is not so much redundant tissue as achalasia. On examination, the internal orifice need not appear diminished in caliber,

although this may be the case, but it is always fixed. When there is no other explanation for inability to void or for persistence of residual urine with good detrusor activity, transurethral resection is indicated and will usually result in satisfactory function. The resection should be circumferential, in effect a lysis, and only a small amount of tissue need be removed, usually no more than 5 gm. It is better to remove too little than too much, and repeat the procedure if necessary. Inclusion of the anterior lip insures division of any striated muscle fibers in that area which might have been subject to spastic contraction. In an experience extending over 16 years, transurethral resection was found necessary in about one out of every four patients during the first three-quarters of that period. At present it is indicated with less than half that frequency.

The striated muscles constituting the external sphincter, including fibers found in the bladder neck, may participate in the spasm commonly observed in areas innervated from levels caudad to the spinal cord lesion. Various methods of sphincterometry have been described, and the results of such studies may be informative or equivocal. Electromyography may be helpful. At times the diagnosis can be established only by therapeutic test. Blocking the pudendal nerves will result in relaxation of spasm and withdrawal of the reflex detrusor inhibition with which it is associated. The effect may last longer than the anticipated duration of the drug's action; if subsequent blocks are undertaken it may be possible to prolong the intervals between them. When the effect is definite but transitory, surgical division of the pudendal nerves may be considered.[6]

Spasm of the larger muscle groups of the abdomen and lower extremities may interfere with bladder conditioning by setting off the detrusor prematurely, so that it becomes impossible to establish consistent function. Since severe spasm of this type is itself a serious impediment to rehabilitation, treatment by anterior rhizotomy or subarachnoid alcohol block is indicated. The former is the procedure of choice if reflex bladder function seems otherwise satisfactory; it may also spare sexual function. Alcohol block will almost invariably relieve the skeletal spasm, at the same time converting an upper motor neuron into a lower motor neuron dysfunction of the bladder, and abolishing erections.[18, 19]

Marked hypertonicity of the bladder, with or without actual contracture, may obstruct all attempts at conditioning or re-training. The

application of a topical anesthetic to the mucosa often brings relief, but this is only temporary. (The apparently paradoxical effect of the same procedure in correcting retention has already been mentioned.) Anti-cholinergic or ganglionic blocking agents are frequently useful. In extreme cases, interruption of the sacral nerves, by blocking or surgical division, may be required. All these methods are also applicable to controlling the autonomic hyperreflexia associated with over-active bladders in patients with cervical or high thoracic lesions.[7, 20, 21]

Occasionally the bladder remains hypotonic. This is the prevalent pattern of the classical "cord bladder" of tabes dorsalis or with other lesions involving the afferent pathways. The reasons for its appearance in the group here discussed are not always clear, but, whatever they may be, the detrusor is inadequate and the anticipated return of reflex function does not occur. Treatment with parasympathomimetic drugs has been disappointing. Stimulation with ice water will almost invariably provoke a response, but the effect is transitory. In some patients with this condition, voiding has occurred during a trial after a prolonged period of tidal drainage, the reason for the ultimate return of function being as obscure as that for delay. Since it is undesirable to confine these people to bed for any longer than is absolutely necessary, the tidal drainage apparatus is connected only at night during such prolonged use, the catheter being clamped during the day and opened at suitable intervals.

In the absence of any other reason for failure to achieve satisfactory function, there is a temptation to charge the patient with lack of cooperation. This is a dangerous refuge; it may develop, after months or years, that the fellow who seemed uncooperative had something wrong with him that we were unable to recognize. Most of these people are anxious to do what they can to help themselves, provided they are adequately instructed and confident of our concern. Yet there are, undoubtedly, a few who cannot or will not understand what is expected of them.

The program for the prevention of renal calculi deserves passing mention, for it is simple but effective. It includes a high fluid intake (3,500 to 4,000 ml. daily), acidulation of the urine, muscular activity to avoid mobilization of calcium from the skeleton, and adequate drainage. On such a regimen the incidence of primary renal calculus should be less than 2 per cent. The weeks immediately after injury are most

critical. Bladder calculi occur mainly as encrustations around the catheter and seem to be characteristic of individual patients rather than specific pathological situations. None of the variously recommended chemical remedies has been particularly effective in my experience, despite some enthusiastic reports. Irrigation with "M" solution (in association with tidal drainage) probably retards the calcareous depositions as well as any other single measure. "G" solution is perhaps more effective but too irritating for general use. Frequent catheter changes are important and it may be desirable to use a straight type instead of a Foley. Definitive hard calculi are less frequently seen than shells, although they may form upon the latter in neglected cases. Both are almost entirely limited to patients still on catheter drainage. They scarcely ever develop in the bladder with good reflex function.

In recent years the diversion of urine through an ileal conduit has been advocated as a treatment for neurogenic vesical dysfunction. There is probably a place for this operation among these patients, but it is a small one. The same applies to other types of diversion or to the formation of a substitute bladder by any means. Such procedures should be reserved for cases in which the bladder is irretrievably and irrevocably damaged and has become a liability. They are not an alternative to corrective management but a last resort when it has failed. Disease of the ureter, with subsequent reflux, hydroureter, hydronephrosis, and renal failure, is one of the complications of neurogenic vesical dysfunction and, as such, beyond the scope of this discussion. But it is entirely appropriate to emphasize here that any new implantation of the ureter should be undertaken only after the most searching evaluation of its capacity for function with its new receptacle. The operation may seem to offer a quick and simple way to resolve a trying situation, but we should be distrustful of short cuts. There is no place for mere expediency in our attack on this problem. Nothing but our best and most painstaking efforts will suffice if the aims we seek are to be achieved.

The system of management just described is not easy. It demands unremitting daily attention to detail, unending explanations to nurses, attendants, and, in particular, to other doctors. It requires the understanding and cooperation of the patient. Other approaches may seem to be easier on all concerned. The only reason for doing it this way—the hard way—is that so far it has given the majority of patients—and

an increasing majority as the years pass—more satisfactory function, better urinary tracts, and longer lives.

REFERENCES

1. Bors, E. Neurogenic bladder, *Urol. Survey 7*:177-250, 1957.
2. Denny-Brown, D. and Robertson, E. G. On the physiology of micturition, *Brain 56*:149-91, 1933.
3. Hunter, DeW. T., Jr. New concept of urinary bladder musculature, *J. Urol. 71*:695-704, 1954.
4. Hinman, F., Jr. and others. Vesical physiology demonstrated by cineradiography and serial roentgenography, *Radiology 62*:713-19, 1954.
5. Bors, E., Comarr, A. E. and Reingold, I. M. Striated muscle fibers of the vesical neck, *J. Urol. 72*:191-96, 1954.
6. Bors, E. and Comarr, A. E. Effect of pudendal nerve operations on the neurogenic bladder, *J. Urol. 72*:666-70, 1954.
7. Talbot, H. S. Management of the spastic bladder in paraplegia, *J. Urol. 79*: 759-66, 1958.
8. Bors, E. H. and Blinn, K. A. Spinal reflex activity from the vesical mucosa in paraplegic patients, *Arch. Neur. Psych. 78*:339-54, 1957.
9. Gould, D. W., Hsieh, A. C. L. and Tinckler, L. F. Behaviour of the intact ureter in dogs, rabbits and rats, J. *Physiol. 129*:436-47, 1955.
10. Talbot, H. S. Role of ureter in pathogenesis of ascending pyelonephritis, *J.A.M.A. 168*:1595-1603 (Nov. 28), 1958.
11. Nesbit, R. M. and Lapides, J. Bladder tonus in spinal shock, *J. Urol. 59*:726-32, 1948.
12. Talbot, H. S. Extraprostatic factors in urinary retention, *New Engl. J. Med. 251*:420-25 (Sept. 9), 1954.
13. Desautels, R. E. and Harrison, J. H. Mismanagement of the urethral catheter, *Med. Clin. N. Amer. 43*:1573-84, 1959.
14. Talbot, H. S., Mahoney, E. M. and Jaffee, S. R. Effects of prolonged urethral catheterization: 1. Persistence of normal renal structure and function, *J. Urol. 81*:138-45, 1959.
15. Nathan, P. W. Sensations associated with micturition, *Brit. J. Urol. 28*:126-31, 1956.
16. Nathan, P. W. Awareness of bladder filling with divided sensory tract, J. *Neurol. Neurosurg. Psychiat. 19*:101-05, 1956.
17. Menzies, R. Conditioned vasomotor responses in human subjects, *J. Psychol. 4*:75-120, 1937.
18. Munro, D. Rehabilitation of patients totally paralyzed below waist, with special reference to making them ambulatory and capable of earning their living: Anterior rhizotomy for spastic paraplegia, *New Eng. J. Med. 233*:453-61 (Oct. 18), 1945.
19. Bors, E., Comarr, A. E. and Moulton, S. H. Role of nerve blocks in management of traumatic cord bladders: Spinal anesthesia subarachnoid alcohol injections, pudendal nerve anesthesia and vesical neck anesthesia, *J. Urol. 63*: 653-66, 1950.
20. Kurnick, N. B. Autonomic hyperreflexia and its control in patients with spinal cord lesions, *Ann. Intern. Med. 44*:678-86, 1956.
21. Meirowsky, A. M., Scheibert, C. D. and Hinchey, T. R. Studies on the sacral reflex arc in paraplegia: I. Response of the bladder to surgical elimination of sacral nerve impulses by rhizotomy, *J. Neurosurg. 7*:33-38, 1950.